THE ETIOLOGY OF HYDRONEPHROSIS.

BY FAXTON E. GARDNER, M.D.,

OF NEW YORK CITY.

THE description of hydronephrosis in classical treatises gives hardly that satisfactory impression of unity one feels after reading a well known and coördinated chapter of nosology. It throws together a number of pathological conditions having but few common features. The list of causes is most heterogeneous: including, as it does, *malformations,* such as imperforations of the urinary tract, stenoses, kinks and torsions of the ureter; *compressions,* depending on an infinite variety of lesions ranging from embryonic remnants and abnormal arteries to tuberculous glands, with the prolapsed, gravid, fibromatous or cancerous uterus, and newgrowths of the bladder and prostate as most frequent causes; *obstructions,* by calculi, by tumors, *traumas, kidney mobility,* etc. And when the tedious enumeration is over, then comes the damaging admission that there are still other cases, the etiology of which is as yet unknown.

All these affections have but one anatomical substratum in common, namely, aseptic distention of the kidney; in all other respects, they differ widely. The pathology is not the same; nor is it even sometimes comparable between cases; for instance, what resemblance between a renal distention due to an imperforation of the urethra and one ascribed to the crossing of the ureter by an anomalous blood-vessel?

The evolution is different: the gravity varies greatly and so do also naturally therapeutical indications.

This wholesale linking of all distentions of urinary ducts by an amicrobian liquid, whatever may be the primordial cause of the trouble, has no advantages whatsoever. It has, on the contrary, numerous drawbacks, especially from the therapeutical standpoint. How can we consider fruitfully in a same paragraph such radically different operations as the removal of a uterine fibroid, the extraction of an impacted stone, and a plastic operation on the pelvis and kidney?

Such a medley is, however, bound to stay as long as text-books give the following simple and elastic definition of hydronephrosis: A distention of the pelvis and kidney by aseptic urine. This definition is incomplete and misleading, because too comprehensive in one way, and too narrow in the other.

It is too comprehensive. Of the two requisites of a good definition it fills only one,—it applies *toto definito* but not *solo definito*: all hydronephroses are renal aseptic distentions, but not all sterile distentions of the kidney deserve the name of hydronephrosis, or else the chapter on hydronephrosis becomes a mere chapter on general pathology.

It is too narrow, because it would lead us to believe that only those collections containing *urine* may be classified as hydronephroses. The contents of a true hydronephrosis are undoubtedly urine at first, but only for a limited time; in old cases the chemical composition is markedly changed; the liquid may become bloody, flaky, turbid, and the condition still be a genuine hydronephrosis.

For the sake of clearness, it is desirable to split the group of affections hitherto described as hydronephroses, and to try to delimit the class of cases to which we might rightfully restrict the name. This is not very easy to do, but when it is done, we find that we have separated a most important group, showing some homogeneity in the etiology, the clinical appearance, and also the treatment.

The specific difference of the cases of aseptic renal distention we want to bring together under the name of hydronephrosis, is that in those cases said renal, or rather renopelvic, distention is a disease *in itself* and not simply an accessory and secondary phenomenon in the course of another disease.

If we apply this principle, first we strike out all those cases in which the distention of the kidney is merely a part of a generalized distention of the urinary tract. For instance, in stenoses of the urethra, renal distention is a relatively common occurrence, if the obstacle to urinary flow is sufficiently marked. It may occur in acquired strictures, but is more frequent in malformations, valvules of the urethra or septal duplicity of the bladder. Then the obstructive agent acts through vesical retention, the distention is bilateral and progresses gradually upwards and finally reaches the kidney. Is it logical to call hydronephroses and consider as a special disease this last stage, this ultimate result, of vesico-uretero-pelvis distention? . . . .

Let us also put aside all cases in which the urinary distention, though localized to the upper urinary tract, perhaps even to one ureter and one kidney, is only an epiphenomenon, a complication, in the evolution of an altogether different disease. In this class of facts, we place many of the compressions, such as those exerted by pelvic or gynæcological tumors. In many cases, as in carcinoma of the uterus, of the prostate, the vagina, the rectum, renal distention is only accessory, overshadowed as it is by the extreme gravity of the primary affection. In other instances, as in fibroids, procidentia uteri, ovarian cysts, or in those rare cases of renal distention caused by peritoneal exudates or tuberculous glands, the mutual relations of the cause and the resulting hydronephrosis are not exactly the same; the condition of the kidney may acquire a very high prognostic significance, and change an operation for the relief of the above-mentioned lesions from a harmless procedure into a very hazardous enterprise.

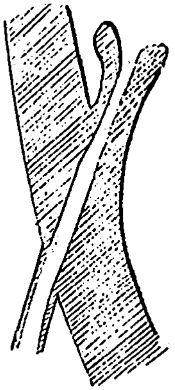
When we have thus cleared the field of a tremendous amount of material, exceedingly interesting indeed from many standpoints, but which must be sorted out under several other pathological headings, where it rightfully belongs, what is left? A class of cases in which the renopelvic distention is the chief feature of the disease, the dominating and essential element. *Anatomically*, the distention is limited to the pelvis and calyces, though sometimes it affects the ureter also, but the dilatation of the latter canal remains accessory. *Etiologically*, all those cases result from non-malignant alterations of the ureter, more particularly defects of its upper segment.

Symptomatically, they are all characterized by a tumor, in the clinical sense of the word, said tumor varying considerably in size. *From the standpoint of prognosis,* they are all benign; *therapeutically* and, last, they all require an intervention on the urinary tract. Consequently, we find between those cases enough similarity to feel justified in uniting them in one group. To that group we restrict the name hydronephrosis.

The narrowing of the scope of hydronephrosis will naturally cause a parallel shortening of the etiological list.

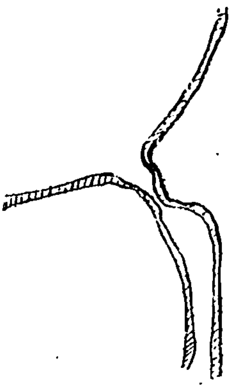
Of the two classes established by a time-honored division, viz., congenital and acquired, the latter class is much the heavier loser; it loses all pelvic benign tumors, all malignant new-growths and a few rarer causes of compression, while the congenital class loses only the hydronephroses due to an imperfection of the lower urinary tract, which are comparatively unfrequent.

FIG. 1.



Congenital malformation (Gerster).

FIG. 2.



Congenital stricture (Moynihan).

But, aside of this limitation, the class of acquired hydronephroses is doomed to lose many more cases which will change sides and array themselves in the rival class. For instance, many of the hydronephroses, formerly attributed to kidney mobility, are now justly ascribed to congenital defects of the upper part of the ureter. Again, hydronephroses caused by vascular anomalies of the kidney must be switched to the congenital group, though their lot has been heretofore cast with the other.

Thus, the class of congenital hydronephroses which, fifteen years ago, was still deemed the lesser and was supposed to contain only pathological curiosities, towers to-day above the other; and each dismemberment of the latter enriches it.

It is not intended to give here an exhaustive study of all possible causes of hydronephrosis. We propose to dwell longer on some of the disputed points, insofar as it can throw some light on that all-important and final aspect of all medical problems, namely, treatment.

CONGENITAL HYDRONEPHROSES.

All these are caused by a congenital imperfection of the ureter. Such an imperfection is an abnormality. Therefore, the best start in a study of the question is a review of the development of the ureter. We shall expose here whatever is needed for a good comprehension of our subject.

The ureter springs as a dorsal evagination of the caudal end of the Wolffian duct. Said evagination ascends in the mesoderm and, while ascending, ramifies. Its offshoots form the pelvis and part of the kidney. Whether they form the whole of the latter, or, what is most likely, only the excretory part, the secretory elements being derived from another origin, is immaterial here; but a movement the ureter executes, or seems to execute, around the Wolffian duct, is very interesting. The ureter is first on the dorsal side of the duct; it successively becomes dorsal and external and finally ventral. The amplitude of the rotation is about 180°. Kuppfer denies that there is any real rotation, because, while the ureter seems to revolve around the Wolffian duct, the distance between the two is constantly increasing. But we must note that in this movement the vesical end stays fixed and that the upper extremity alone moves and therefore the duct cannot but become twisted around its axis. This rotation must be borne in mind because its rôle in the production of some hydronephroses is certain, though variously estimated.

Much more important and less hypothetical are the facts we are going to consider now. At the end of the third month of intra-uterine life, the excretory organs have assumed their final shape; the primary and intermediate kidneys, pro- and mesonephros, and their excretory canal, the Wolffian duct, have vanished and henceforth urinary secretion, due allowance being made as to the quantity, is comparable to adult secretion. But the ureter is not then the smooth and rather uniform canal dissection shows in full-grown subjects. Its outline is exceedingly uneven: constricted segments follow dilated ones, and valvules formed either by the mucosa alone or by a folding-in of the whole wall of the canal, abound. These irregularities are not equally distributed in all points: their most common location is near the renal pelvis; next comes the segment near the bladder and, last, the middle portion; in one word, their elective sites are both terminal parts of the ureter.

Wölffler was the first to draw the attention to these points. In 100 newborns, he found 20 times more or less marked permanent transversal folds of the mucosa. In all of the 20 cases there were some valvules near the pelvic ureteral outlet, in a few cases some were found besides at a distance varying from a few millimetres to 1.5 centimetres from the vesical meatus. In ten instances, the jutting parts were prominent annular valvules, scarcely letting a filiform catheter pass through the duct. Said valvules, according to Wölffler, were formed simply by the mucosa.

But Wölffler's researches had not begun early enough. If, instead of studying newborns, he had examined fœtuses, he would have been brought to look upon those valvules, not as frequent anomalies occurring once in every five cases, but as constant temporary formations in the development of all ureters. This was ably brought to light by English. If we examine a lengthwise section of the whole duct, we notice the following peculiarities: the pyelo-ureteral junction is always a narrow point; the immediately underjacent segment shows always a number of plicatures formed by a folding-in of the whole muscularis. The mucosa covers both sides of the valvule, but the external coat takes no part in its constitution; at the implantation point, the latter bridges over the gap between the two layers of muscularis constituting the valvule and the space thus delimited is filled up with connective tissue.

The number and size of those formations vary; sometimes single, sometimes multiple and placed in tiers one above the other along a short segment of the canal; sometimes low transversal ridges, sometimes real movable valvules, the free edge of which is tossed up and down by the urinary flow. Their implantation on the walls of the ureter is on a somewhat spiral line; hence the serpentine aspect of the canal. At the base of the fold, a little furrow may be seen on the outer coat and, here also, the superposition of these furrows makes the ureter appear as though it were spirally twisted around its axis. The valvules greatly reduce the lumen of the duct, sometimes to such an extent as to prevent catheterization with a hair. Pulling on both ends of the ureter causes the folds to flatten for the time being, but, as soon as the traction ceases, they reform, provided the outer coat has not been torn by too rough a pulling.

Besides these mucomuscular valvules, purely mucous formations are also found, particularly in the juxtapelvic segment of the ureter; some of these are vertical and radiate up into the pelvis where they array themselves in star-like figures. On a few of these mucous valvules without muscularis, papillary raisings have been found.

These three kinds of formations are *normal,*—we desire to lay stress upon the fact. They are always observed in the same part of the ureter, the rest showing none. They exist on both sides and are even almost always homologous; if, however, there is only a narrowing of the lumen on one side, valvules are found on the other. The plicatures take in both the muscularis and the mucosa, but not the outer coat, which fact precludes the idea of acquired lesions; for in the latter eventuality, all three coats would be involved.

At the end of the third month of intra-uterine life, the ureters are thin cords tapering from the lower to the upper end with two markedly distinct segments; the lower is wider and grows faster than the upper which has no distinct lumen, filled up, as it is, by vertical mucous folds, and mucous, and chiefly mucomuscular, valvules.

All those projections disappear under the action of two factors: the eccentric pressure of the urine and the normal growth of the canal. When, during the fourth month of fœtal life, urine begins to flow through the ureter, the plicatures flatten, the narrow points widen, and the calibre becomes even. And at the same time, the length of the ureter increases markedly. The lower segment growing quicker than the upper, valvules persist where both sections meet, said meeting point corresponding to the pelvic ureteral outlet in the adult. Thus is the most frequent location of anomalies in the juxtapelvic segment accounted for.

We easily conceive that, if normal ureteral growth is impeded by any cause, valvules will persist. The growth of the ureter being influenced by the same factors as the growth of the kidney, we readily understand why abnormalities of the kidney and of the renal blood-vessels are so frequently associated with imperfections of the ureter. An arrest of development of any part of the reno-ureteral system cannot but have its *"contre coup"* on the other parts.

Thus we account for the frequency of hydronephrosis in ectopic, movable, single, and horseshoe kidneys. This explanation is much more logical than the change of direction of the ureter generally given as the reason of retention in horseshoe kidneys. As for movable kidneys, the question is more complex and will receive due attention later; many movable kidneys are congenital; but some are not.

The two above-mentioned influences work still after birth and tend to perfect the shaping of the ureter almost up to adult life. Newborn infants and young children still present a variety of slight ureteral defects, while in full-grown subjects all pelves show a fairly uniform type.

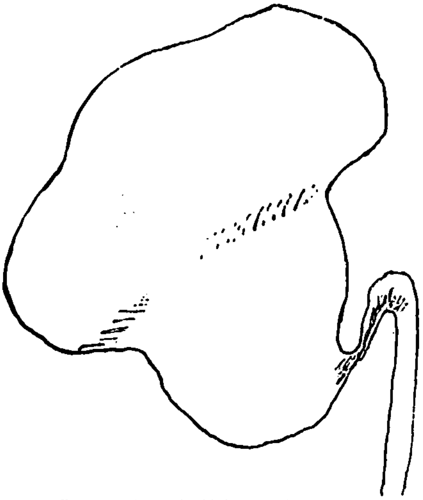
After this summary of the development of the ureter, how can we but think that in the persistency of one of those fœtal dispositions lies the cause of most congenital hydronephroses? The constancy during intra-uterine life, the frequency at birth, are weighty arguments and there is ample evidence to prove that the so-called normal strictures of the ureter are traces of those transitory folds.

Let us now enumerate briefly the different classical causes of congenital hydronephrosis. It is wrong to make *absence* and *multiplicity* of the ureter head the list; for neither can be a cause of hydronephrosis. We have already stated that no hydronephrosis is possible when the ureter is absent. The two or three cases on record of hydronephrosis supposedly belonging to that type are old cases and are probably based on an erroneous interpretation of facts.

Nor can *multiplicity* be a cause of retention. I do not know of any more fascinating study for those who love to delve into embryological puzzles, than that of the development of supernumerary ureters; but it would lead us too far from our path and it is only indirectly connected with our chief subject. Enough is it to say that the anomalous ureter represents, most likely, in women, the Wolffian duct, in men, the Müllerian duct. This accounts for the common termination of said ureter in the utriculus prostaticus in men, in the upper vault of the vagina in women. It may be easily understood that such an important anomaly is frequently accompanied by defects in the calibre, particularly by atresia of the lower opening. Then hydronephrosis develops; but when the diameter is *normal*, a supernumerary ureter cannot cause any retention. It may give rise to a special variety of incontinence; but in a majority of cases, it does not give rise to any symptoms whatever, and many a man has lived a long life without his anomaly being even suspected. In all but one of the published cases of partial hydronephrosis due to a stenosed supernumerary ureter, the latter drained the upper part of the kidney, which upper part alone was distended (Rémy, etc.); Hochenegg's case is the one exception.

The preceding remarks apply integrally to *abnormal endings* of the ureter, the next cause we find on the classical list. Not the abnormal ending provokes hydronephrosis, but the frequently concomitant atresia, or the modifications the anomaly brings in the direction of the ureter: flattening of the duct, sharp bends, etc. . . . . In Walter's case, the ureter opened too high into the bladder and consequently its intraparietal course was too obliquely ascending. In a case of Weigert's the opening on top of the verumontanum was very narrow and the corresponding kidney was a large uronephrosis. Needless it is to multiply examples; they are all alike.[1]

FIG. 3.



Pseudo-stric ure by kink and adhesions.

Kinks and *torsion* of the ureter are also causes of retrodistention of the kidney. Thomson, Roberts and Arnould explain kinks by the embryonal rotation of the ureter around the Wolffian duct. This explanation has generally been held as valid without discussion, but, when carefully scrutinized, it does not seem as illuminating as at first sight. Kinks are much more naturally explained by the persistency of the normal fœtal condition. What we have said above dispenses us from dwelling more on the point. But the rotation theory explains very well the torsions that are sometimes observed. We mean here *real* torsion, not the torsion so many authors have considered as a cause of hydronephrosis, but which is only an apparent, a *pseudo*-torsion. Real torsion is rare; we could find but four cases in the literature: the last one was published two years ago by Dr. Grégoire, who showed us the specimen. The ureter was plainly twisted around its axis, and the pressure of the liquid within the sac was at times sufficient to untwist it; hence intermittent evacuations. But specimens of that type are rather unique. What is commonly described as a torsion of the ureter is only a peculiar aspect due to the spiroid furrows that mark the implantation of valvules on the ureteral wall, where the outer coat bridges over the tucked-in muscularis. The same thing is well known in the cystic duct where Heister's valvules give a deceptive spiroid appearance while there is no real torsion. And furthermore we do not see very well how the ureter, attached to the kidney above, to the bladder below, can become twisted around its vertical axis. Torsion would be possible only if the kidney revolved also around its vertical axis. Hamonic has admitted the existence of such a displacement, but without quoting any definite examples, and he grants it is only slight; it must be so, because the renal pedicle would soon oppose it. In a case where no renal pedicle existed, Schede saw a real complete torsion (360°).

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[1] Special mention must be made of *intravesical prolapse* of the lower end of the ureter, which is not exceptional, and is sometimes associated with hydronephrosis of the corresponding kidney.

Anomalies in the calibre are much less frequently mentioned in clinical reports, and, nevertheless, they certainly constitute by far the most important group of causes of hydronephrosis. That this is due to the greater difficulty to detect them is evident, and that we see only what we have learnt to see is a thread bare truth. They will be found in a very large number of cases, and have already been found whenever they were carefully looked for. *Valvules* are the most frequent, as might be naturally inferred from the development of the ureter. But there may be also plain *strictures* without valvules. We have drawn in Fig. 2 such a case, borrowed from Moynihan; the ureter was narrowed as if it had been tied with a tape. Fig. 1 represents a peculiar deformity seen by Dr. Gerster. Matthews Duncan and Carpenter have published similar cases. This stricture near the pelvi-ureteral junction seems simply an exaggeration of a normal anatomical condition: Bazy on his mouldings found it very often, and Byron Robinson, Poirier and others, have shown that that point is always, even in normal ureters, one of the tight passages.

Embryonal remnants of the Wolffian duct (Launay) or of the Müllerian duct (Reliquet and Mathias-Duval) are rare and deserve no more than a passing notice.

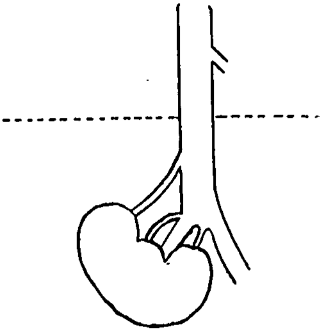
To the group of congenital retentions must be added unhesitatingly many of the cases formerly ascribed to kidney mobility, and hitherto classed among acquired hydronephroses.

Kidney mobility is in most cases congenital. Figs. 4 to 7 show the four types of movable kidneys to which all others may be reduced. In the first type, the kidney is plainly ectopic, its blood-vessels are abnormal and have an abnormal origin. In the second, the movable kidney, disposed either vertically or obliquely, may sometimes be pushed upwards up to the lumbar fossa, but the origin of its vessels is still abnormal. In the third type, the kidney is vertical, the pedicle is much elongated and the vessels spring normally from the vascular trunks, while in the fourth the pedicle is short and the kidney oblique.

The two first types are unmistakably congenital; the third is also, while the fourth represents the typical acquired movable kidney. Consequently, if hydronephrosis develops on account of kidney mobility in a kidney of any of the three first classes, it is a congenital hydronephrosis.

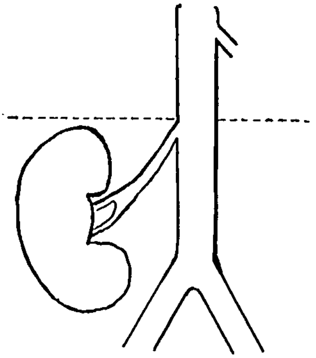
But this is not the only reason pleading for the change we are now considering. There is another, more important, namely, that many of the retentions ascribed to kidney mobility *are altogether irrelevant of the latter* and are indeed due to some of those congenital defects the capital rôle of which we have already seen.

FIG. 4.



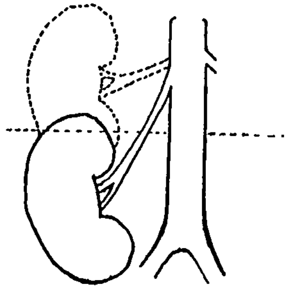
(Pasteau).

FIG. 5.



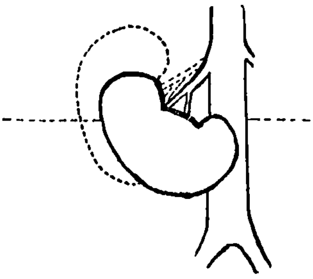
(Pasteau).

FIG. 6.



(Pasteau).

FIG. 7.



(Pasteau).

Different types of movable kidneys.

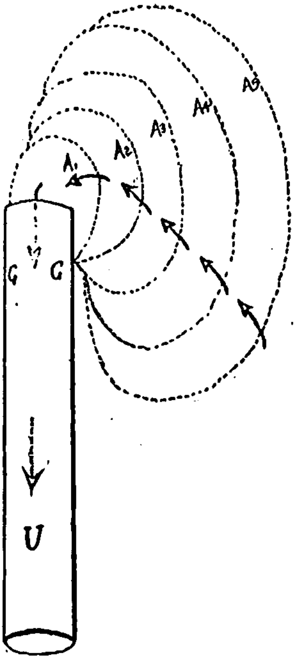
Such a view, very few years ago, would have been revolutionary, but it is now fast gaining recognition. Since Landau's series of papers and since Terrier and Baudouin's work, it has been almost universally taken for granted, and believed almost as a tenet, that movable kidneys cause the type of hydronephrosis called intermittent.

The latter is characterized essentially by crises of pain, accompanied by a progressively developing renal tumor, and by temporary oliguria; it ends by a more or less abundant evacuation of urine, while the tumor subsides. A very simple explanation is obvious. When the kidney falls, the ureter kinks, the flow of urine is mechanically stopped, the urine is retained in the kidney, hence the tumor and the oliguria; then later, the obstruction is removed, the accumulated urine is voided, hence the polyuria and the disappearance of the tumor.

Unfortunately for medical truth, this explanation is too simple, too elegant, too logical and therefore too alluring. It seemingly accounts perfectly for everything and makes further investigation seem superfluous. Here we have intermittent symptoms and an intermittent cause, the chronology of which seems to tally nicely together. How could one not be the cause of the other? And if difficulties arise when it comes to deciding as to the mechanism, many ingenious hypotheses can be brought forward and we can even describe more or less imaginary anatomical peculiarities to give our hypotheses a solid substratum. All this has been done in good faith, but nevertheless it is forcing facts into the frame of a theory, instead of enlarging the frame to make it fit the facts, or even casting it off, if the latter adaptation is not possible. The parallelism between renal displacements and intermittent hydronephrosis cannot stand a close scrutiny.

First of all, there is no special type of disease to which the name intermittent hydronephrosis properly belongs. Intermittence is not a characteristic of a *special* kind of retention; it is a common feature of all retentions; and is presented by all at some period of their development. *Hydronephrosis is not simply a distention of the kidney;* if it were, it might be truly intermittent; but hydronephrosis is more than that; it is a distention with *progressive sclerosis* of the pelvic walls and of the kidney. Now the latter lesions are permanent. Consequently, what is intermittent is not the hydronephrosis, but only the filling and emptying of the sac. Now this has but a secondary importance and is not sufficient ground to establish a distinct type.

FIG. 8.



(Fenger).

Again, the polyuria which comes after the attack of pain is very often out of proportion with the size of the tumor. In some cases, the excess of urine voided over the normal amount reaches almost a gallon, while the tumor is comparatively small and certainly cannot contain such a quantity of liquid. Inversely, catheterization during the crisis has found sometimes not more than *one ounce* in the pelvis. This shows conclusively that the supposed relationship between the filling and emptying of the sac and the growth and subsiding of the tumor, are only apparent. The polyuria is not due to the sudden evacuation of a renal retention. The increase in the size of the kidney is due in many cases to an intense congestion of the organ; when the congestion ceases, polyuria sets in, and the volume of the gland falls again to normal. The postcongestive hyperactivity of all organs is a well-known fact.

Beyond any doubt, many of the hydronephroses associated with kidney mobility are congenital, and are due to one of those defects we have already mentioned several times. We should prefer to believe that kidney mobility and hydronephrosis are completely independent from one another. Both are congenital defects: they are simply co-existant: hydronephrosis does not create mobility, any more than mobility creates hydronephrosis. We simply have to deal with an hydronephrosis *in* a movable kidney.

But is there no room left for hydronephrosis caused *by* kidney mobility? Extremists say no; but we think there is on the question a vast amount of clinical and experimental material which we have no right to disregard. No doubt it would be more satisfactory to the mind, always intent on uniformity, if it were proved that all hydronephroses associated with kidney mobility are in fact congenital hydronephroses, but this we do not believe to be rigidly exact. Certainly, retentions due to kidney mobility must be reduced enormously in number, but they must not be wiped out entirely. The point will be dwelt on later.

An argumentation much along the same lines applies to those cases of hydronephrosis commonly ascribed to pressure of an abnormal blood-vessel on the ureter. If such a pressure begets hydronephroses, the latter must be termed congenital, although, in text-books, they are generally classified among acquired.

In some cases of hydronephrosis, the ureter has been found, near its origin, astride on an anomalous blood-vessel, an artery in most cases, a vein in a few exceptional instances. This is enough to have the question of causality raised.

The solution is not evident at first sight; the vessel may be the cause of the retention, but it may also have nothing to do with it, and simply have assumed connections with the sac secondarily as a consequence of the growth of the latter. In the literature cases may be found demonstrating both eventualities.

In all cases, the blood-vessel is *abnormal.* Anatomy tells us about several arteries which may come into contact with the first segment of the ureter. First, the lowest branch of the renal artery which goes to the lower pole of the kidney may separate prematurely and pass in front of the origin of the duct. Next, supernumerary arteries branching off directly from the aorta; in one out of every four cases, the vessel passes behind the ureter, in the three others it passes in front. Thus, in a vast majority of cases, the anomalous artery crosses the ureter *anteriorly.*

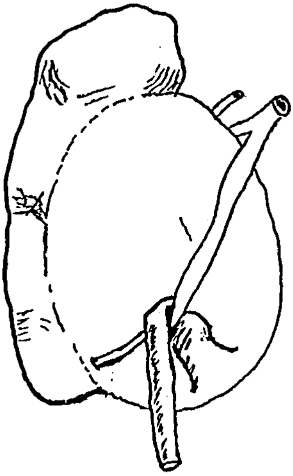
Can such an artery cause hydronephrosis? The older writers admitted it unreservedly; three mechanisms have been put forward: (*a*) intermittent compression by the arterial systolic impulse, (*b*) continuous compression as if by a fibrous tract, and secondary alterations of the canal, and (*c*) more particularly, kinking of the ureter on the vascular cord, determining either a sharp bend or a more or less open curve. Boogard, who observed the first case in 1857, thought even there might be in those cases a *true* intermittent hydronephrosis, the liquid accumulating till its pressure becomes high enough to overcome the arterial obstacle, and then emptying.

But this is not a matter of course, though apparently simple, and it is bitterly contested now.

English was the first to state that an abnormal artery passing *in front* of the ureter cannot possibly be the cause of a hydronephrosis, because it can never hook the ureter of a movable kidney. If the artery passes *behind* the duct, then it may hook it when the kidney falls. This conception would reduce greatly the number of cases.

Küster, Israël, Bazy, Duval and Grégoire, concur. They even reject altogether the rôle of abnormal vessels in the production of renal retentions. As in several cases the presence of a ureteral malformation is expressly noted (Roberts, Morris, Decressac), they contend that *all* hydronephroses developed in kidneys showing an abnormal blood-distribution are due to such defects. They assert that *because* of the uronephrosis does the vessel come into contact with the ureter. Of course such a theory is tenable; kidney mobility, abnormal vessels and ureteral defects belong all three to the same class of errors of development and, when one exists, the chances are that some of the others exist also. But it is partly hypothetical and we think it is carrying the desire for uniformity too far.

Fig. 9.



(Decressac).

From Bazy's own drawings, purporting to prove that a pre-ureteral vessel could not produce a retention, while a retro-ureteral vessel could, Pierre Delbet drew diagonally opposed conclusions. And a peculiarity noted several times sustains the idea of hydronephroses *by* and not simply *with* abnormal vessels. Figs. 9 to 12 represent cases of this type. It may be easily seen that in 9 the dilatation of the ureter does not begin exactly where the artery passes over the duct. In some cases, the dilatation begins above the crossing point, in some cases it extends below. It is plain the artery is not then the cause of the retention. But, in some others, the crossing point marks exactly the beginning of the sac as in Figs. 10, 11, 12. Some authors account for this by saying that the crossing point is precisely the part of the ureter where congenital imperfections are most often met with, but is not this straining a point too far? When they assert that, in those cases, before proclaiming the primordial rôle of the artery, we must prove that there is not in the ureter a malformation, they forget that the burden of proof rests upon them and that *they* have to establish the rights of their claim. We, therefore, admit with the older authors, and with Legueu, Hartmann, Luys, Picqué and Carlier, the existence of hydronephrosis *by* vascular anomaly. Those cases are not very numerous, but they exist, and they raise a very interesting therapeutical problem.

ACQUIRED HYDRONEPHROSES.

This group as described by classics is the larger, but also the more heterogeneous, of the two. Our definition of hydronephrosis splits it and, with compression cases, takes away more than half of its clinical field. Furthermore, it has been steadily decreasing with the inroads of congenital lesions in its domain; it is now very much reduced, but it is not as near evanescence as some would have us think. It still takes in a very respectable number of cases dependent on the following factors: acquired movable kidney, cicatricial lesions of the ureter, impacted stones, and more rarely inflammatory changes of the duct, ureteritis and peri-ureteritis.

This list is not long when compared to the older ones, but it is sufficient to give acquired hydronephrosis an honorable place in nosology.

Acquired movable kidney is a cause of hydronephrosis, though we have already seen that it is by no means as frequent a cause as has been contended at a time.

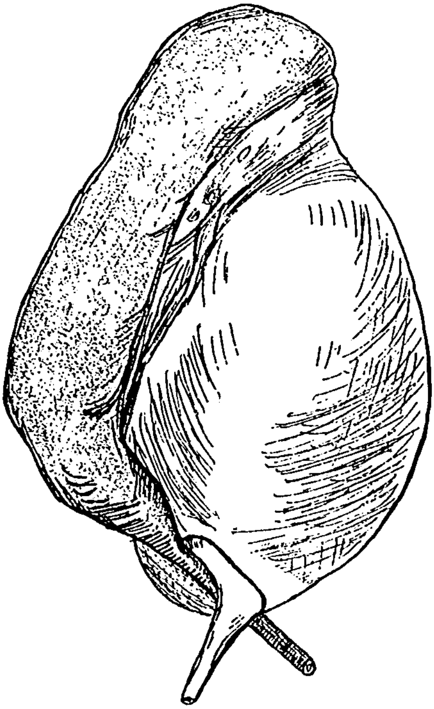
Experimentally, Tuffier succeeded five times out of nine in producing hydronephrosis by making the kidney movable. But there is one requisite well demonstrated by Legueu, Hildebrandt and Haga: that the upper ureter *be fixed.* If it is not, no hydronephrosis can be obtained. Paul Delbet was so impressed by the latter fact as to view ureteropyelitis as the only real cause of hydronephrosis; but his, like all extreme theories, contains some truth, but overshoots the mark, and gives too much weight to a single etiologic factor.

If kidney mobility may under some circumstances provoke hydronephrosis in animals, why should it not do it in man? All agree that a slight obstacle to urinary flow is enough to cause a retention. Why deny the same right to kinks of the ureter, constant in cases of movable kidney? Ureteral catheterization shows the presence in the pelvis of simple movable kidneys of small retentions varying from two drachms to an ounce or more. May we not rightfully consider these as the starting point of larger accumulations? Those who want to find at any cost a congenital malformation at the origin of every hydronephrosis do not let such minor facts nettle them. They say those small retentions are not hydronephroses, because they lack an essential element,—namely, sclerosis of the walls of the sac. But do not even congenital hydronephroses go at first through an initial stage when sclerosis of the walls has not yet begun? A retention does not, from the outset, contain a pint of liquid; it must begin by a few drachms or an ounce.

Willing, as we are, to grant that many hydronephroses hitherto ascribed to kidney mobility, are really due to congenital defects; that movable kidney is itself, in a great majority of cases, a congenital disease, we still stoutly defend the existence of acquired hydronephroses, caused solely by the mobility of an acquired movable kidney.

Wounds of the ureter are not common, but the resulting scars are so constituted that the risk of retrodistention is great. Judging from the nature of the healing process, stenosis seems an unavoidable contingency and experimental and clinical surgery of the ureter gives corroborative evidence. In the scar, the anatomical elements are atrophied and lost in a dense connective tissue which forms almost the whole of the ureteral wall and even, in some cases, extends considerably beyond, and gives rise to diffuse sclerous peri-ureteritis, thus greatly aggravating the stricture.

FIG. 10.



(Luys).

A wound of the ureter may be produced from within by a stone; we shall have to discuss later calculous hydronephrosis. It may also be produced by a cutting or pointed instrument; but, in the latter case, hydronephrosis is an exceptional occurrence. A urinary fistula is a much more common sequel; among hydronephroses, we found but one case, Soller's, concerning a man who was struck in the lumbar region in 1870, during the Franco-German war, by a splinter of a shell, and at whose necropsy, ten years later, a stricture of the left ureter with a large hydronephrosis was found.

Most cases of acquired strictures of the ureter are observed after *deep contusions of the loins.* This brings us to discuss the question of *traumatic hydronephrosis,* a very moot question which, however, seems to be nearing a definite solution.

Most of the cases described as traumatic hydronephroses may be summarized thus: an individual, a man or a child in most instances, falls from a height, the lumbar region striking against a hard prominence; or gets run over by a vehicle, a wheel of which passes diagonally in the costo-iliac space. Right after the accident, the usual symptoms of renal contusion are present: more or less severe pain, shock, hæmaturia. A few days or a few weeks later a large tumor is felt, extending horizontally from the side to the median line, vertically from the hypochondrium to the iliac fossa. Said tumor is *not* heralded in, we insist on the fact, *by either pain or a change in the general condition* of the patient. When tapped, it yields a considerable amount of a yellow, transparent, urine-like liquid. After the tapping, the tumor forms anew and it becomes necessary to thrust the needle again, three, four, or more times before a cure is effected.

Such is the history of the princeps case reported by Hawkins in 1834; such are the more recent reports of Stanley, Hicks, Vincent, Cropt, Cabot, Nové, Josserant, Froelich and others.

On such a foundation, some writers build a clinical type denominated traumatic hydronephrosis and establish a theory to explain how it is brought about. They take it for granted that the accumulated fluid is really *within* the renal cavities.

The obstacle to urinary excretion is peri-ureteral hæmatoma pressing on the duct. The above-jacent portion of the ureter dilates and kinks and thus the obstruction becomes complete.

But, supposing this theory had an anatomical confirmation, which it has not, we would still wonder how such a rapid and large dilatation of the pelvis and kidney could be possible. Here we have a most unusual feature; never has such a distention as is noted in the clinical reports, been produced in so short a time. Clinically, it takes years before a large hydronephrosis is constituted; experimentally, renal distention follows ligation of the ureter, but never do experimental tumors attain such a huge size. Many searchers have ligated the ureter; I have done it myself several times in the course of experimental work directed along other lines, but I have never seen anything approaching the dimensions of the so-called traumatic hydronephroses.

Another fact deserves attention. These enormous distentions of the pelvis, if such they be, develop without pain. Oftentimes they are detected only because surgeons, after a renal contusion, are wont to palpate the injured region. Now, let us catheterize a ureter and inject some liquid in the pelvis and distend it, ever so little; before five seconds, the patient will be howling and writhing. I have seen it many times. Sinitzine and Albarran have demonstrated the fact long ago. Rapid distention of the pelvis is extremely painful; and some would have us believe that enormous hydronephroses can develop painlessly in a few days! And also that those enormous hydronephroses may be cured by simple tapping!

Therefore we find so far in the theory of traumatic hydronephrosis a mere hypothesis lacking anatomical support and presenting several weak points in opposition with well established facts observed time and time again.

Now, what do direct anatomical findings teach us? Hawkins, whose christening of his first case as hydronephrosis was responsible for the birth of the above-exposed doctrine, states himself expressly that, in his case, the volume of the kidney was *normal;* that the flexuous and elongated ureter inserted *normally* on the pelvis and had no connections whatever with the cyst containing the fluid; said cyst being *sub-*

peritoneal and *pararenal,* but altogether *independent* from the kidney. Is this a hydronephrosis? It might have been considered as such in 1834 when the terminology was lax, but nobody would keep the name to-day, not even with subcapsular as a corrective. The same independence of the kidney and ureter from the collection is noted in all cases of traumatic hydronephroses which came to operation or necropsy. This, coupled with some other findings, and the nature of the fluid, will give us the key of the problem.

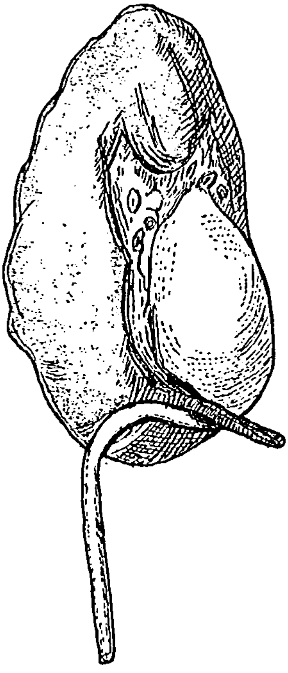
The finding just alluded to is the frequent injury of the pelvis and ureter. Two tears existed in the pelvis in Hawkins' case, one in Vincent's. Barker found a hole in the ureter and Chaput saw the latter completely torn off. The kidney itself is, as a rule, uninjured, and this accounts for the infrequency of hæmaturia much better than the weak reasons given by the partisans of the clot theory.

The liquid withdrawn by tapping is *urine;* of course, not exactly normal urine, but nevertheless easily recognizable as such.

Let us now simply enumerate the three above-mentioned characteristics and we shall reach the true definition of these post-traumatic tumors: an *extrarenal* collection *of urine,* produced by an *injury of the pelvis or ureter.* Now we grasp at once the why of the painlessness and of the quick growth of these tumors. Here we have a normal kidney, secreting normally, and pouring its secretion into loose connective tissue in which a pouch may be readily excavated. While studying the effects of puncture on the pelvis and ureter, we had many occasions to see how painlessly the largest collections develop without any effect on the general condition.

That the acute, almost sudden, development of a genuine hydronephrosis, after an injury, is impossible, is our firm belief. However, there are scattered in the literature a certain number of cases in which the truly hydronephrotic nature of the tumor is undeniable. Thus Schede says he saw a tumor appear six days after the accident: an operation and a post-mortem showed it was a true hydronephrosis. Barling's case is similar; it occurred a few days after the patient had slipped while getting into his bed. Fenger's patient had made a violent effort dismounting from horseback; and, last but not least, ten days were enough for a hydronephrosis to appear in one of Richardson's patients after the trifling exertion caused by the rolling of a barrel.

FIG. 11.



(Luys).

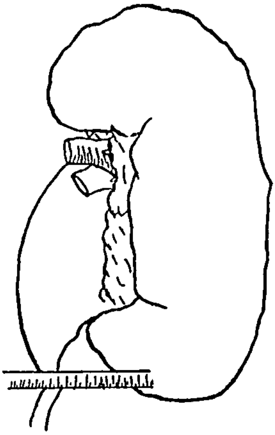
The *post hoc, ergo propter hoc,* character of these deductions is obvious. These cases must be construed as cases of latent hydronephrosis discovered because a trauma drew the attention to the loins. The slightness of the injury in many instances makes us suspicious as to its etiological rôle.

But is there nothing that may be called traumatic hydronephrosis and must we scratch the name from medical nomenclature? We do not think so. If a lumbar contusion cannot produce hydronephrosis itself, it can very well cause a stricture of the ureter. The symptoms of hydronephrosis will appear after a certain interval, four months, six months, several years. The interval was six months in Dr. Gerster's remarkable case which enjoys the distinction of pertaining all at once to a traumatic stricture, to a congenital malformation, to one of the first conservative attempts ever made, and also to the youngest patient on record among those treated by conservation; unfortunately after six months' success, a sinus reopened, pyelonephritis set in and nephrectomy had to be performed. Shall we say, with some writers, that the latter facts are not *traumatic hydronephroses,* but should be styled *hydronephroses secondary to a traumatic stricture* of the ureter? This might be a subtle distinction, if it were more than a mere quibble. If it was sustained, we could call congenital only those cases in which the tumor is fully developed at the time of birth; all others would have to be styled hydronephroses secondary to a congenital lesion of the ureter. Calling, as we do, congenital, all lesions relevant of a congenital condition whatever may be the time of onset of the symptoms, we must call traumatic all retentions caused by a trauma, even if the latter acts only indirectly and tardily through the medium of a stricture. And, to sum up tersely the etiologic rôle of injury in the genesis of hydronephroses, let us say: *immediate traumatic hydronephrosis cannot and does not exist, late traumatic hydronephrosis does exist.*

There have been many ups and downs in the history of *calculous hydronephrosis.* In turns, it has been considered without sufficient grounds as very frequent, and its existence has been almost flatly denied.

When a rather large sized stone is found impacted in the ureter while the corresponding kidney is dilated, a natural enough inference is that the calculus is the cause of the hydronephrosis; it was the opinion of all the earlier writers, Rayer, Morris, and was still admitted without dispute by Newman in 1888. The stone was supposed to act either as a valve or a plug when its size made it unmovable. It is very likely that many hydronephroses described as calculous on the sole strength of the co-existence of an impacted stone, do not deserve the name; but much of the criticism aimed at calculous hydronephroses seems to bear upon secondary questions. Thus Arnould rejects all cases in which the content of the sac is not exactly like normal urine. Such an exclusion is arbitrary, because we know the liquid contained in a hydronephrosis may become reddish, flaky, even turbid, and yet remain sterile. Legueu refuses the name of calculous hydronephrosis to all cases in which bacteriological tests have not peremptorily established the perfect asepsis of the contents. This also, although logical, is arbitrary; such a stringent rule may be theoretically right, but practically the difference between a strictly aseptic sac and the same when it is slightly infected, is not so great as will warrant placing those two stages of the same condition in two different classes. There are no such sharp lines in biology. Such requisites cut down Newman's 51 cases to 2 (Antona, Stevenson). But neither Arnould nor Legueu enters into the real merits of the question: can a stone produce an aseptic distention of the kidney, and, if it does, how

FIG. 12.



(Legüeü).

does it act? They argue on contingencies and their ostracism is as far from scientific truth as the first observers' hasty conclusions.

It is impossible to deny that a stone impacted in the ureter can produce hydronephrosis. Clinically, the causal relation is proved by the cure of retentions after simple removal of the calculus. Experimentally, Ebstein and Nicolaïer produced renal calculi by feeding oxamid to dogs and Tuffier and Navarro, while using the method with other ends in view, obtained incidentally a few retentions.

But the number of calculous hydronephroses, though much higher than thought by Arnould and Legueu, remains nevertheless small as compared with the number of renal and ureteral stones. Hydronephrosis is not the ordinary result of stone impaction in the ureter; atrophy and sclerosis of the kidney is much more frequent, as the general pathology of duct calculi would lead us to think, and as every surgeon who has occasions to see many cases of aseptic lithiasis knows. Therefore, some peculiarities are necessary in order to have a stone cause a retention. Careful analysis of the cases elicits a few of them; but sometimes nothing out of the ordinary is found. For those cases, the old theories are alone acceptable and why should not they be accepted since a partial obstruction is admitted by all surgeons as the only necessary cause for the production of hydronephrosis?

However, there are here no fast rules governing the development of retentions. Neither the shape or volume, nor the number, nor the seat of the concretions, are decisive elements. Hydronephrosis is sometimes seen with stones of the size of a filbert; while there was no retention in Israël's case with a huge ureteral calculus seven inches long and more than one inch in diameter. One stone is enough in many cases, while 16 in a case published by Morris had not caused the pelvis to dilate. Why there should be such variations is not always discernible.

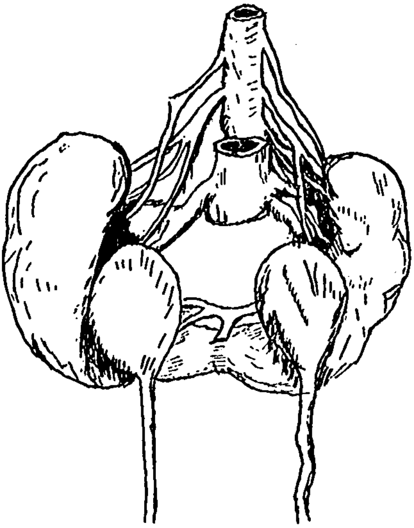
In some reports, the co-existence of a congenital malformation is noted (Fenger, Perkins, Morris, three cases of Léon Bernard). It is more than likely that stone and malformation helped each other constitute an obstacle, while each one taken separately might perhaps not have impeded the flow of urine.

In some other instances, a similar aggravating rôle is played by acquired alterations of the canal. When a stone moves through the ureter, it certainly scratches the walls, and more or less deep traumatic lesions are a natural consequence, despite the statement of many writers that calculi are harmless for the duct through which they are expelled. But in most cases, these lesions heal quickly and well. Sometimes, however, a stricture is the result; when it is formed the next stone will stop there and a sufficient obstruction will be realized. Barker, Israël, Ledentu, Albarran, have seen cases of this type. Bernard has seen, in newborn infants, four calculous hydronephroses caused, not by common stones, but by deposits of uratic sand in the kidney; and he raises the question if some of the supposedly causeless hydronephroses in adults are not sequelæ of an infantile undiagnosed lithiasis. But it is noteworthy that, in three out of his four cases, there were malformations of the ureter and maybe, after all, his were congenital hydronephroses also.

The popping in and out of the stone has been proposed by Lancereaux as an explanation for some cases of intermittent hydronephrosis. It reminds one of an old-time theory of recurrent appendicitis based also on intermittent poppings of fecal concretions. However ingenious the idea, it does not stand examination. We have said that there was no special type deserving the name of intermittent hydronephrosis; consequently we need not dig out of our imagination mechanisms to account for its production.

Inflammatory lesions of the ureter do not rank as frequent causes of hydronephrosis for the very plain reason that if the ureter is infected, the chances are that the pelvis and kidney are also; pyelo-nephritis or pyonephrosis, not hydronephrosis, is the logical outcome. But, if the infective process is slow and very much attenuated, hydronephrosis seems to be a bare possibility. *Ascending tuberculous ureteritis* has been incriminated; but cases remain few and far between. To our knowledge, four cases only are on record, all French (Lancereaux, Albarran, Répin, Legueu). The ureter is sometimes still pervious, but, in the majority of cases, it is occluded. And maybe the name hydronephrosis does not fit exactly those cases; not unlikely, they are healed tuberculous kidneys and distention is only the trace of the old disease. They depend more on destruction of the parenchyma than on distention of the pelvis, and consequently are pseudohydronephroses, akin to renal atrophy more than to real hydronephrosis.

Fig. 13.



Horseshoe kidney. Double uronephrosis. (Hauser).

Peri-ureteritis is more important as an etiologic factor. It is very frequent in the upper part, where it changes temporary bends into permanent kinks. It has been stated above that, without the fixity of the upper segment of the ureter, no hydronephrosis could result from kidney mobility. In the lower part, peri-ureteritis is not as frequent, but it is observed sometimes: the duct, encircled in a hard fibrous ring is narrowed and possibly deviated from its normal course; the destruction of the band and the straightening of the ureter are needed for a cure of the retention.

Here ends our list of the causes of hydronephroses. The official list still includes curves, kinks, flexuosities, torsions, acquired valvules, oblique and high insertion of the ureter, but these are direct *consequences* of the pelvic distention and not its causes. So thought already Simon (of Heidelberg), the father of renal surgery, and his opinion has been vindicated by Albarran and Legueu's experimental work. All those who have ligated ureters know that the first effect of ligation is a sagging of the upper part of the ureter and the development of flexuosities. Fenger has taught us how the high and oblique insertion of the ureter is a consequence of the development of the retention, as well as the valvule near the ureteral opening, which, it must be noted here, is thoroughly different from a congenital valvule. (See Fenger's diagram, Fig. 8.) Torsion has already been referred to.

Such are the views on etiology of hydronephrosis we have derived from the observation of numerous enough cases, which it was our good fortune to see, and from painstaking study of the literature on the subject. Maybe we do not show as exclusive preferences in the choice of our theories as most writers. But what is most needed in the study of the etiology of hydronephrosis is good judgment and impartiality. Strange to say, almost all writers seem to have extreme opinions. Fifteen years ago, almost all hydronephroses were considered as acquired; since the frequency of congenital defects began to be recognized, the wind has shifted and many writers seem now bent on demonstrating that all are congenital, while some few remain staunch uncompromising supporters of the old belief. One way is as unscientific as the other. A theory, even a pet one, ought to always remain subservient to facts. Facts show conclusively that the etiology of hydronephrosis cannot be reduced to the new slogan, "arrest of development," any more than it could be to the old, "kidney mobility." Truth lies midway between extremes, "*Suum cuique.*"